and not much information which can be considered to be directly relevant to Pharmaceutical Engineering: thus the book is slightly misleading.

There are 3 chapters based on ^{1}H-NMR of proteins, two describing conformational changes of staphylococcal nuclease and one centred on antibody combining sites. The studies outlined in these articles are based on 'state of the art' techniques and are quite readable. The chapter outlining the structure of the nicotinic acetylcholine receptor is particularly well referenced and provides a precise overview of this important membrane-bound protein.

The book contains a useful group of chapters which describe how site-specific mutagenesis is capable of facilitating our understanding of enzyme mechanism. Studies based on aspartate aminotransferase, trypanothione reductase, triosephosphate isomerase and aspartate transcarbamoylase, together with two chapters describing enzyme mimicks (cyclodextrins and immunoglobulins) form the core of the book. Aspects of quarternary structure, ligand/substrate binding sites, detailed kinetics, and transition state analogues are presented in some detail.

There is only one chapter entirely devoted to bacterial expression, purification and characterisation of a protein; namely the HIV1 protease. Nevertheless, this isolated study is useful as it provides details of methods capable of isolating the enzyme in 'reagent quantities', purified to homogeneity.

The publication is relatively free of errors, is generally well referenced, containing many post 1986 citations and is reasonably priced. The subject matter will be of more interest to protein chemists and enzymologists than to pharmaceutical scientists.

R.C. Hider

Reviews on Cancer: A Special Issue on the Human Immunodeficiency Virus; Edited by W.J.W. Morrow; Biochimica et Biophysica Acta, Vol. 989, No. 3, Elsevier, 1989; vi + 92 pages; $27.50

This welcome update on the AIDS research situation put together for BBA by W.J.W. Morrow of IDEC Pharmaceuticals takes the form of a series of 6 papers from leading groups covering the current clinical and social situation, all aspects of the HIV infection and life cycle process, the immune response to the virus and recent attempts at vaccine development. In 1988, a set of AIDS reviews was published in Scientific American. At that time author's views, which no doubt reflected the collective feelings in the scientific community, were essentially upbeat and optimistic. The massive research effort underway would in the end prevail. After all this was not anything as intractable as cancer; merely an infectious disease with a clearly defined and cultureable agent. This assurance was bolstered by the detailed knowledge then uncovered of the virus structure, genetic and physical, and its life cycle. Many of the early questions had been answered and on the basis of evidence available plausible hypotheses had been advanced for the rest.

The feeling pervading this BBA issue is more uncertain and pessimistic. Depressingly little movement towards a cure can be detected in the intervening period. In addition, many of those assumptions about the way HIVs 1 and 2 go about their fatal business have not been validated. In fact several have been partially undermined.

Take the straight forward notion that the AIDS condition is caused by HIV attacking and destroying the CD4-bearing T-helper lymphocytes, a view fostered by the experimentally verified fact that the virus binds to and infects any cell sporting these receptor molecules at their surface via a direct interaction with CD4. However, as discussed in Sattentau's article, although the population of T-helper cells declines during the course of the disease, very few in fact carry the virus. Also, cells which do not make CD4 are now known to be infected. How does HIV get into these? Kieber-Emmons, Jameson and Morrow in their review on the role of the gp120-CD4 interface report that mutations can be inserted into the viral coat protein which do not affect its ability to bind to CD4 but which do destroy virus infectivity. Does HIV therefore have a second cell surface ligand and is this more important for the net pathological effect? These new questions are challenging the old dogma and the picture is less certain than before.

The fact that patients die because they cannot defend themselves against secondary infections is obvious. But is this only due to the lack of an adequate number of T-helper cells? In their paper, Evans and Levy describe evidence which demonstrates a reduced ability of circulating natural killer cells and of aberrant functioning of phagocytic cells in AIDS patients. Volberding and McCutchan in their stimulating overview of medical aspects which introduces the series describe data indicating that the opportunistic infection to which patients are susceptible very much depends upon the original route of infection with HIV – a strange finding if only T-helper cells are involved.

Perhaps the least gloomy of the collection is the paper by Kieber-Emmons et al. dealing with the structural, immunological and pathological facts relating to virus-receptor interaction. Here there have been important developments. Soluble CD4, which is the extracellular portion of this membrane glycoprotein, has been shown to inhibit viral infection in vitro. When tested in vivo however it was quickly cleared from the blood stream. Genetic manipulation was applied to the problem and a soluble CD4-immunoglobulin heavy chain chimera was produced. This survives for much longer in the circulation of patients and is currently under clinical trial. The results are eagerly awaited for this test will reveal whether or not the currently held image of the disease has feet of clay. If HIV pathology is really mediated by an alternative and as yet unknown cell surface receptor, then soluble CD4 will have little effect on the progress of AIDS in patients.

One serious difference between this and the earlier series is the presence of an article in the latter on available drugs and strategies for the development of new ones to interfere with viral reproduction and spread. In the BBA collection there are only 2 paragraphs on chemotherapeutic aspects and only AZT is mentioned. This is surely a major omission, difficult to understand in light of the readable vaccine review by Zhou

and Kennedy which concluded the booklet and revealed little progress in this much researched area. Possibly the author(s) concerned did not make the deadline?

Of course this volume, like many Elsevier publications, is far too expensive to buy personally, but it is well worth a visit in the library - that is if your library can still afford to take BBA at £3051 per annum.

C.J. Chesterton

The Molecular Biology of Alzheimer's Disease; Edited by Caleb E. Finch and Peter Davies; Cold Spring Harbor Laboratory; Cold Spring Harbor, New York, 1988; xii + 197 pages; $25.00 (paperbook)

'The Molecular Biology of Alzheimer's Disease' is the latest in the series Current Communications in Molecular Biology published by Cold Spring Harbor Laboratory. This volume of less than 200 pages is based on the proceedings of the Banbury meeting held in 1988 and covers the current state of understanding of Alzheimer's Disease at the molecular level.

Alzheimer's Disease (AD) is a brain-specific disease that results in severe cognitive impairment and is characterized by an abundance of neuritic plaques and neurofibrillary tangles and the selective degeneration of nerve cells located mainly in the cerebral cortex and hippocampus. The condition, its possible causes and physiochemical manifestations are summarized in 30 short chapters grouped into 7 major themes; (i) the molecular biology of the amyloid fibril percursor protein, (ii) the characterization of paired helical filaments, (iii) animal models and species specificity for AD, (iv) regional specificity of the pathology, (v) degeneration and neuroplasticity, (vi) non-neuronal involvement in AD pathology, and (vii) genetics of AD. Molecular handles for the study of AD are the accumulation of A4 protein and neurofibrillary tangles, and a major proportion of the book concerns the role of the A4, or beta protein, in AD. Possible explanations of the accumulation of the protein fragment are discussed in relation to the fact that the same protein is found abundantly in Down's syndrome brain (trisomy 21) and the gene for the A4 precursor is located on chromosome 21. The presence of paired helical filaments may be a further example of the accumulation of aberrant polypeptides particularly as they appear to be flagged for potential destruction by their conjugation with ubiquitin. Gene dosage, control of gene expression and protein catabolism are areas probed as causative factors for these changes in brain protein biochemistry. The presence of a protease inhibitor sequence on the A4 precursor is suggestive that proteolytic functions may be important in AD. Scrapie with the 'infective' protein or prion and mouse trisomy 16 are presented as possible models of neurodegeneration. However it remains to be clarified whether the accumulated aberrant polypeptides so characteristic of neurodegeneration are the direct causal agents of AD, or whether these apparently incompletely degraded polypeptides are non-pathological symptoms of an unidentified neurotoxic factor which also provokes their accumulation.

A.R. Hipkiss

Transmembrane Signalling, Intracellular Messengers and Implications for Drug Development; Edited by Stefan R. Nahorski; John Wiley, Chichester, 1990; xvi + 248 pages; £39.50

As an overfrequent participant at scientific meetings, I do not care to have the repayment of my fare or accomodation being made conditional on the delivery of a script. I sense that many others who delight in presenting their work and ideas for discussion at meetings share my dissatisfaction with a form of publication which is generally inaccessible to most readers (i.e. rarely do these appear in a sequence of a serial publication), almost invariably out of date (two years is not unusual) and very expensive. There are exceptions of course and on occasion I have been happy to arrive at important meetings or exotic places with my ticket underwritten by a paper. Similarly, I can imagine most of those invited to speak at the annual Biological Council Symposium on Drug Action in the Spring of 1989 were also happy to arrive, script in hand. The reason for this is that the meeting was held in the magnificent 18th century lecture theatre at the Royal Institution near Piccadilly in London, made famous in earlier times by such as Humphrey Davy, Michael Faraday and John ('Blue Sky') Tyndall (and which should not to be confused with the Royal Society whose facilities pale in comparison).

So, what have we got? The volume at just under 250 pages comprises 14 chapters and encompasses most of the main areas of interest expressed in the title. Mainly this means the mechanisms by which the concentrations of cyclic AMP and Ca^{2+} inside cells are regulated and appropriately the consideration of subsequent events (e.g. regulation of protein phosphorylation and dephosphorylation) is excluded. There is nothing on retinal transduction and there are a few other obvious omissions. A number of leading laboratories are represented by authorship and doubtless anyone working in the area will find something of interest. More than this, a number of the chapters are well introduced with nice historical sections (e.g. Regan, Caron and Lefkowitz on adrenergic receptors, Buckley on muscarinic receptors). At a time when all is being swept aside by cloning techniques, I suspect that these introductory words will have more staying power than anything else and in this respect the articles will serve those students who require a historical background. The best